REMARKS

Independent Claims 1, 19 and 36 and dependent claim 39 have been amended to more clearly state the invention. Claims 5 and 31-35 have been canceled, without prejudice. Support for the amendments made herein can be found in the instant specification, for example at [0054]. It is believed that none of these amendments constitute new matter and their entry is requested.

Rejections under 35 U.S.C. § 112

The Examiner has rejected claims 1, 3-6, 19 and 31-40 under 35 U.S.C. 112, first paragraph, as failing to comply with the enablement requirement. Applicant respectfully disagrees and traverses this rejection. Cancellation of claims 5 and 32-25 obviate the rejection of these claims. As discussed below in detail, the specification provides evidence demonstrating that levels of peptides of SEQ ID NOs: 1 and 4 directly correlate with the presence of the magnesium binding defect. Thus, detection of the presence of these peptides provides a method to detect the magnesium binding defect and predict the risk of developing associated disorders, such as preeclampsia.

The Specification Provides Evidence that a Lower that Normal Level of Peptides of SEQ ID NOs: 1 and 4 Correlates with the Magnesium Binding Defect

The prior art established that some component of plasma from a normal (normotensive) rat model, which component was present at lower levels or missing in plasma from hypertensive rat models, could ameliorate the magnesium binding defect and reduce blood pressure otherwise observed in the hypertensive rat models. [0042] The instant application reports Applicant's discovery of components found in normal plasma (e.g., plasma from normotensive rats) which are at lower levels in plasma from hypertensive rat models. The results reported support that the levels of peptides of SEQ ID NO.1 and 4 (the "Peptides") in plasma membranes of hypertensive

rats are lower than normal. Specifically, the results demonstrate that increasing the level of the Peptides yielded magnesium binding at normal levels in the hypertensive rats, yet had no significant affect on magnesium binding in plasma membranes of the normal normotensive rats. See Example 2 and Table 2.

The above conclusion is based on the following: 1) Normal serum levels of Peptides are presumed present in normotensive rats which do not have the magnesium binding defect; and 2) administration of Peptides to hypertensive rats increases the serum levels of Peptides. As such, we know that the serum levels of Peptides were lower when the magnesium binding defect was observed, and the levels were higher after administration of Peptides when magnesium binding increased to normal levels in the hypertensive rats.

In summary, the instant specification provides sufficient teaching to enable one skilled in the art to compare and correlate the levels of Peptides with the magnesium binding defect. Direct measurement of the Peptides is within the ordinary level of skill in the art. Furthermore, the specification provides evidence of the biological activity of the Peptides, i.e., that increasing the level of the Peptides increases magnesium binding up to normal levels, and concomitantly ameliorates the magnesium binding defect. Thus, the specification provides a reasonable correlation between detecting the levels of Peptides and identifying individuals having the magnesium binding defect.

The Examiner has rejected claims 19 and 31-35 under 35 U.S.C. 112, first paragraph, as non-enabling for the full scope of the claims. The cancellation of claims 31-35 and amendment herein of claim 19 obviates this rejection.

In view of the foregoing amendments and remarks, Applicant urges that the instant specification enables the full breadth of the claims and requests that the rejections under 35

U.S.C. 112, first paragraph, be withdrawn. If the examiner is of the view that any issue remains unresolved, it is respectfully suggested that Applicant's undersigned attorney may be contacted by telephone at the number set forth below.

Respectfully submitted,

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